Amendments to the specification:

Please amend the title on the cover sheet of the application and on page 1, line 3 as follows.

~~RPS GENE FAMILY, PRIMERS, PROBES, AND DETECTION METHODS~~
METHODS OF IDENTIFYING PLANT DISEASE-RESISTANCE GENES

Please amend the table beginning on page 31, line 23, as follows.

Table 1

Mutant	Wild type	position of Mutation*	Change
rps2-101C	703 TGA 705	704	TAA Stop Codon
rps2-101N	1741 GTG 1743	1741	GTGGAGTTGTATG Insertion (SEQ ID NO: 216)
rps2-102C	1426 AGA 1428 arg	1427	AAA amino acid 476 lys
rps2-201C	2002 ACC 2004 thr	2002	CCC Amino acid pro

- Nucleotide positions refer to SEQ ID NO:215

Please amend the paragraph beginning on page 40, line 5, as follows.

Finally, inspection of the *RPS2* sequence reveals a fourth *RPS2* motif, a potential membrane-spanning domain located at amino acids 340-360. Within this region, a conserved GLPLAL (SEQ ID NO:217) motif is found at amino acids 347-352. The presence of the membrane-spanning domain raises the possibility that the *RPS2* protein is membrane localized, with the N-terminal leucine zipper and P-loop domains residing together on the opposite side of the membrane from the LRR region. An orientation in which the C-terminal LRR domain is extracellular is suggested by the fact that five of the six potential N-linked glycosylation sites occur C-terminal to the proposed membrane-spanning domain, as well as by the overall more positive charge of the N-terminal amino acid residues (see, e.g., Kornfield et al., supra; von Heijne, supra). A number of proteins that contain LRRs are postulated or known to be membrane-spanning receptors in which the LRRs are displayed extracellularly as a ligand-binding domain (see, e.g., Lopez et al., Proc. Natl. Acad. Sci. 84:5615 (1987); Braun et al., EMBO J. 10:1885 (1991); Schneider et al., supra).